Clean Copy of Amended Specification Page 1
Pursuant to 37 C.F.R. § 1.121(b)(1)(ii)

METHODS FOR DECREASING BETA AMYLOID PROTEIN

Cross-Reference to Related Applications

This application is a divisional of pending prior application U.S. Serial No. 09/239,387 filed January 28, 1999, which is a divisional of U.S. Serial No. 09/046,235 filed March 23, 1998, now U.S. Patent No. 6,080,778. The United States government has certain rights in this invention by virtue of National Institutes of Health grant number RO1NS33325 to Bruce A. Yankner.

Background of the Invention

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Alzheimer's disease (AD) is the most common cause of dementia in the aged population. The accumulation of large numbers of senile plaques containing the 40-42 amino acid amyloid β protein (Aβ) is a classic pathological feature of AD. Both genetic and cell biological findings suggest that the accumulation of Aβ in the brain is the likely cause of AD (Yankner, B.A. (1996) Neuron 16, 921-932.; Selkoe, D.J. Science 275, 630-631 (1997)). Strong genetic evidence in support of the pathogenic role of Aβ came from the observation that individuals who inherit mutations in the amyloid precursor protein almost invariably develop AD at an early age. These mutations increase the production of a long variant of the Aβ peptide that forms senile plaques in the brain (Goate et al., (1991) Nature 349, 704-706). Mutations and allelic variations in other genes that cause AD, including the presenilins and apolipoprotein E, also result in increased production or deposition of the Aβ peptide. Reiman, et al. (1996) N.E.J.Med. 334, 752-758, reported that in middle age, early to mid 50's, individuals who are homozygous for the Apo E4 gene have reduced glucose metabolism in the same regions of the brain as in patients with Alzheimer's disease. These findings suggest that the pathological changes